ART. VII.—*On the Nature of Phthisis Pulmonalis, and especially of its First or Pre-Tubercular Stage.* By EDWARD SMITH, M. D., LL. D., L. R. C. P.; Assistant Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; Corresponding Member of the Natural History Society of Montreal, &c.

WHATEVER may be the disadvantages of hospitals devoted to the treatment of a single class of diseases, it must be admitted that they offer the best opportunity for inquiries of a scientific character in relation to those diseases, both by the numbers of such cases brought together at one time and by the concentratedness of attention which an earnest inquirer is led to devote to the subject. Those who have had the good fortune to prosecute a part of their medical studies in France must cherish the remembrance of the facilities for study afforded by the special wards of Louis when at the Hôpital Beaujon, the Clinique d'Accouchement, the Hôpital St. Louis, the Hôpital du Midi, the Bicêtre, and the Hôpital des Enfants malades; and, after a lengthened period, will still revert to them with great readiness and pleasure. I think it an advancing, and not a retrograding spirit, which of late years has caused the establishment in London of various institutions for the treatment of special classes of affections; for, whilst it must be admitted that in such institutions, wisely conducted, particular diseases may be treated as well as it is possible for them to be in other and general institutions, in progress of time it will be found that those special institutions will have led to a better acquaintance with such diseases. Whilst, therefore, I would admit that it is possible to carry the limitation to an unwise extent, I am of opinion that the profession should encourage those which are wisely established and conducted, and that pupils would find it to their advantage, in subsequent practice, to have devoted some small share of their attention to the practice of special hospitals.

Having, at the Hospital for Consumption and Diseases of the Chest, at Brompton, the greatest possible opportunity for the study of chest affections, and particularly of phthisis, I have recently engaged in several series of inquiries as to the nature and treatment of the latter disease; and the conviction has arisen in my own mind, that if we would successfully combat it, it must be in the pre-tubercular state, and that it is possible to determine with sufficient precision the disease at this early period. The belief in the futility of all our efforts in this disease, and therefore of all hospitals devoted to its treatment, is based upon experience derived from treatment at an advanced stage, and from the belief, common as I conceive, that the tubercle is both the essence and commonly the earliest recognizable indication of the affection. If we still remain of this opinion as to the nature and stage of the disease, I do not see how any greater success in our practice can be looked for; but if, on the other hand, there is a recognizable anterior condition—one in which no marked deposit has as yet appeared—then, certainly, we may have sanguine hopes both of preventing and of arresting the disease.

This, then, appears to me to be a subject of the greatest possible importance, and one upon which all our improvements must be based.

The opinion as to the nature of phthisis, which has prevailed for some years, is that it is a blood disease—a disease not merely accompanied, or even preceded by an abnormal condition of the blood; but one originating in, and essentially due to the disease of the blood. Hence, it is regarded as a general disease of the system, with a local manifestation in the lungs, and as allied with scrofulous diseases in which a deposit like to tubercle is also met with. The frequency with which tubercle is said to be found in various parts of the body at the same time, and with which the disease appears in members of the same family, or in the children of consumptive parents, are regarded as circumstances corroborating this view of its blood origin. In explaining the nature of this supposed change in the blood, it is not affirmed that tubercle is met with in that fluid; but the fault is ascribed to what Rokitansky—in a former, but not in the last edition of his work—termed a fibrin crasis, in which the change may be in quantity or quality, but not in either universally.

In combating this theory, I shall not weaken my opposition if I admit that, in some instances, the blood was probably changed from a state of health at the very commencement of phthisis; for my objection to the theory is, that its advocates have fallen into the common error of affirming that to be true of the whole which is true of a part. No one would deny that the state of the blood cannot be the same precisely in two individuals, or in the same individual at every hour of the day, or in succeeding weeks or years, when it is remembered the source from which the blood is derived, its intermittent supply and varying character, as well as the frequent variations in the vital powers of the system. But surely much more than this ought to be required before the blood is thus said to be in a state of disease, and one, moreover, which can induce a special disease in the system locally or generally. To prove the existence of the disease in the blood, and its connection with the local disease, it is certainly needful that elements common and peculiar to the two should be found in it, or at least that by inoculation or some other mode of communication, the diseased blood can originate in other systems the disease said to arise from it. Otherwise it is an unsustained assertion, a mere guess, a mode of reasoning which would not be admitted for a moment in any system of reasoning. Now in this case the tubercle is not found in the blood, the disease is not communicable by inoculation or infection. As to its transmission from parent to child, that must result from the solids as well as the humours, and therefore is not the communication of definite elements, but a state of the whole system which is inordinately prone to the production of the disease. Moreover—and this I think would be fatal to the theory—in a very large proportion of cases the disease has attacked so insidiously that the period of its origin cannot be determined from any state of the health then existing which differed from the ordinary health of the individual. To call that state of the system a state of general disease in which the general health is unchanged, is an absurdity, as it would be also to presume either that the precise elements of disease had existed in the blood during years of health, or that they had suddenly arisen without any cognizable cause otherwise than local, and were then incapable of demonstration. The most that can be affirmed is, that in these cases of supposed blood disease the whole system, both solids and fluids, was inordinately predisposed to the attack of phthisis.

Neither would it weaken my argument if I admitted that, during the disease, the blood may be or even is changed from a state of health; for that would only be valid to the extent of admitting a co-ordinate or a resultant condition. It might show one of the qualities or powers of the disease, but it could not show its essential value; for, by the proposition, the disease had originated before the diseased state of the blood occurred. My objection, here, is that the advocates of the theory have erred in affirming the nature of a thing from its consequence, or possibly only from its coincidence.

The observations of Rokitansky fall under this head; for, in his remarks upon the fibrin crasis of tubercle, he refers only to the state of the blood in the disease; and although he has now withdrawn them, it cannot be supposed that the opinion of one who, for some years, has exerted no inconsiderable influence over our schools and literature, can be so readily forgotten. Hence it may be profitable to see on what unsubstantial grounds he based an argument which has influenced the whole profession. It is worthy of note how indefinite he is compelled to make his opinions, as though he felt that a fallacy was involved somewhere. He says, at the onset: "Although there are, no doubt, tuberculoses purely local." And again: "The latter (meaning the tuberculous dyscrasis) is, for the *most* part, developed out of a fibrin-crasis." Thus he offers two limitations at the outset, beyond which his theory does not apply. He then continues: "In the dead subject, a hyperinosis of the blood may not be ostensible. Nay! owing to the frequent and voluminous processes of exudation attendant upon the course of a tuberculosis, a poverty in fibrin is, perhaps, more likely to reveal itself after death. Excess of fibrin has, however, been demonstrated in such cases by the chemical action of blood withdrawn during life, and it may also be inferred from the extent of tuberculous deposition. *Still the hyperinosis is far from being adequate* to account for the disease. It is the qualitative anomalies of the fibrin that must determine its tuberculous nature—anomalies of quite a peculiar kind, which we shall see may modify every one of the fibrin-crases referred to into the tuberculous."

Thus, having excluded an indefinite number of cases from the influence of fibrin crasis, he admits that the remainder may have either an excess or a deficiency in quantity, and that quantity is far from being adequate to account for the disease. As the nature of a thing must be determined from properties which are universal to it, it follows that, in Rokitansky's showing, the whole value of his argument rests upon the proof which he can adduce as to the quality of the fibrin; for he again says: "Still these said crases by no means serve to throw much light upon the nature of tuberculosis." Then how does he prove the peculiarities in the fibrin which are to give rise to the tubercle? Strange as the answer may be, he takes the characters which he says are exhibited by tubercle, and then simply affirms that they are the peculiarities of the fibrin crasis. Such are the surpassing proneness to deposition and its high grade of coagulability; and in reference to these he gives the following remarkable passage: "The granulation of tuberculous products of inflammation upon serous membranes might indeed be ascribed to a separation of the tuberculous portion due to its coagulability. Still, the very localization of the tubercle crasis in such minute and sharply defined area, that little granule-shaped exudates are the result, constitutes a peculiarity—a remarkable peculiarity—seeing that the same form of separation—the same form of tuberculization—attaches to the fibrin of hemorrhage and to endogenous vascular coagula."

I shall presently refer to an opinion on the origin of tubercle which would have afforded an explanation of the peculiarity now referred to, far more rationally than that now offered; but, for the moment, I content myself with simply asking you to note the multitude of limitations and doubts which he has himself expressed, and to say if truth need be hedged about in this manner.

But upon the broad fact that there is any change in the chemical qualities of the blood, even in (much less at the origin of) the disease, I quote the authority of Lehmann, who distinctly affirms that he could not discover any characters in the blood peculiar to phthisis, but only such as resulted from attendant circumstances, as excess of fibrin during the complication of inflammation, and of water during that of sweating. No doubt changes must occur in the blood during the progress of so exhausting and chronic a disease as phthisis, but they do not in any way affect the question of the origin and the nature of the disease.

But, notwithstanding the preceding remarks, I fully believe that a defective constitution of body (which, I presume, is really all that is commonly meant by the term blood disease) is a frequent cause of phthisis, and as such is directly associated with whatever other conditions will directly or indirectly tend to exhaust the system. Of the two allegations, viz., that as the disease is universal it must arise either from one cause universally existing and acting with something approaching to an equal degree of intensity in all known conditions; or that it arises from many causes, all acting in the human frame in one common way: I unhesitatingly adhere to the latter. The multitude of causes are well known, and accord with universal belief, and the one common mode of attack is, in my opinion, a diminution in the inspiratory action of the air-cells of the lung.

The short limits of a paper do not permit me to enter into this subject fully, and, therefore, I would give my reasons for this belief in the most summary way possible.

If the disease arise from general causes it must be sought for in one of the two great functions of alimentation and respiration taken in their widest sense.

In reference to the former, I have proved by an inquiry at the Hospital for Consumption, &c., and partly published in the *British and Foreign Medico-Chirurgical Review*, for April, 1856, that in the early stages of phthisis the quantity of food taken is equal to that in health, and that it is properly digested. I have also shown that if the excretions, the feces, and the solids in the urine are not greater than in health, that the excretions by the liver and skin in the early stage are probably not greater than in health, and that the sputa is the only external source of waste which at that period can be seized upon. If such be so in the early stages of the disease, *a fortiori*, they would be so at the period of origin of the disease, and hence I infer that the general cause does not lie in the want of food and in the process of digestion.

The following table contains the results of this extensive inquiry, and offers some points worthy of attention:—

TABLE IN OUNCES AVOIRDUPOIS. JUNE.

Showing the Daily Averages of the Ingesta, Egesta, Pulsation and Respiration in Fourteen Male Cases of Phthisis, at the Hospital, derived from Observations made Twice a Day for a Month.

Hospital Nos.	69	95	107	88	79	75	46	80	51	90	58	73	85	77	Total average.
INGESTA:															
Solids	40.1	32.3	21.1	22.3	30.1	28.6	26.6	28.8	14.7	21.7	31.4	15.2	19.7	19.	24.6
Fluids	103.	72.2	83.3	53.	63.3	60.	67.	62.6	92.8	43.6	82.6	62.	54.8	58.3	67.9
Total	143.1	905.5	104.4	75.3	93.4	81.6	93.6	98.2	207.5	65.3	91.4	72.2	74.5	70.3	92.7
EGESTA:															
Feces	7	7.5	7.8	2.84	9.6	5.65	5.82	4.75	5.85	6.94	7.55	2.6	4.82	2.34	6.3
Vomits			1.82		.20										.14
Sputa and saliva	2.2	16.6	9.7	2.8	3.94	.77	5.	0.9	.75	5.2	2.2	5.9	2.	1.73	2.8
Urine	58.5	36.8	50.8	36.2	32.2	42.5	39.8	32.6	64.	21.	49.5	23.9	30.	25.7	38.8
Total	67.7	55.6	62.9	48.4	45.1	48.9	53.	39.2	70.6	32.3	59.5	32.4	36.5	29.7	48.
Excess of ingesta over egesta	75.3	49.5	42.2	33.8	48.2	32.6	43.6	52.9	36.9	33.	64.4	44.8	38.	40.5	44.66
Weight of body (lbs.) monthly	+2.8	+.1	+1.4	—.6	—2.1	+1.1	—.4	+.7	=	+.1	+2.2	+.8	—2.2	+.4	+.6
Solids in urine (grains)	1226	856	776	758	732	704	676	626	604	596	592	574	574	550	70.3
Pulse	84.1	86.4	92.6	91.3	92.7	83.2	969	949	76.2	97.5	89.5	104.6	928.4	88.5	95.3
Respiration	16.	22.6	24.9	28.	20.6	97.	22.7	20.6	22.4	24.7	25.6	31.	20.6	29.7	23.
Meat and fish	6.8	6.2	5.2	5.4	4.1	4.3	3.8	4.7	.5	4.2	5.2	3.9	3.7	4.	4.4
Wine			2.6		8.					9.9		9.4		.9	.5
Ale and porter	15.1	8.2	14.7	19.	8.7	8.	10.	7.4	8.	2.4	8.	9.9	8.	20.	10.
Bread	21.7	18.4	9.1	8.7	14.	9.1	15.6	94.	6.8	8.6	96.6	8.4	7.9	8.2	12.

But in reference to respiration there is a change so marked as to arrest our attention, and so important as to lead to the belief of its being the immediate cause of the mischief.

In the very earliest moment of phthisis it has been already proved that there is a great diminution in the vital capacity of the lung—a diminution vastly too great to be accounted for by the accompanying dulness as indicative of the extent of tubercular deposition. It is true, that with all the help which our art can afford, the latter statement is matter of opinion, but I submit that although it is not capable of the demonstration which now belongs to the former, the conviction on the mind of the investigator is as strong as it could be if it were made cognizable to the eye. At this, as at an earlier period, there is a lessened amount of vesicular murmur and the presence of harsh respiration, and, perhaps, of wavy respiration or of prolonged expiration. The two former indicate a diminution in the action of the air-cells, the first directly and the second indirectly by the absence of the vesicular murmur which, when present, masks the bronchial sounds, and when absent presents them with their clear, short, and harsh characters to be distinguished. The disparity between the amount of diminution in the vital capacity of the lung and the tubercle then existing appears to me to be thus explained: that is, by regarding the lessened action of the air-vesicles with or without the deposited tubercle as the cause of the lessened quantity of air. This view is corroborated by the flattening of the chest, which is so constantly found even at that stage with a marked degree of lessened mobility, and, at the same time, diminished resonance on percussion, either in the slight degree which has been hitherto attributed to a natural dulness on percussion peculiar to certain individuals, or to the further degree indicating the presence of some solid material. The atrophy and the lessened mobility can only result from inaction or a lessened action of the subjacent lung on the principle that any part of the system diminishes in size as its action is lessened. The slightly lessened resonance can only be accounted for by the absence of that which when present gives the clear sound, for the construction of the chest is the same in all persons, except in those who have that part covered with fat or largely developed pectoral muscles, and it is not in either of these cases that the disease in question is commonly found. To these may be yet added another evidence, viz., the short and feeble inspiratory effort which even to the unaided senses is so frequently perceptible, but which by the help of the stethoscope is almost invariably met with in the early stage.

Thus all the circumstances of the case lead us irresistibly to the conclusion that there is greatly lessened action of the lungs, and that to an extent quite beyond the influence of the then deposited tubercle.

But to this statement may be urged the objection which I have already taken, viz., that evidences attending the disease are not necessarily anterior to and causative of it. To meet this, therefore, I would call your attention to the large mass of patients who attend as out-patients at our hospitals, who, by common consent, are believed to be prone to phthisis, and in them we find the same flattening and lessened mobility, the same feebleness and shortness of inspiration, and the same lessened vesicular murmur, all or some of them, but, probably, to a less extent than is found in the early stage of phthisis. At this period, also, I have ascertained by a newly-invented spirometer that the quantity of air admitted into the lungs per minute is less than in healthy persons, and in no small proportion of such persons the natural capacity of the chest is small from a contraction of that cavity in all its diameters.

As the period of origin of phthisis is never ascertained with exactitude, I cannot specifically refer to the physical signs then existing, and as any case in which the signs just mentioned are present may not pass into tubercle, it is scarcely possible to give absolute proof of the connection between these signs and the tubercle of a later stage. Here again, therefore, we must in some degree rely upon common professional belief as to the class of persons predisposed to phthisis; and in such I venture to affirm that some or all of the evidences of lessened respiration now given will be found.

Now to what part of the lung structure does this lessened action apply. Obviously it applies mainly or exclusively to the air-cells. The bronchial tubes are mere conduits and do not directly bring the air into contact with the blood; that duty is reserved for the air-cells alone; but whether the power of expansion of the lungs is not in some degree conferred upon the minutest bronchi may be open to doubt; for I have ascertained that in an advanced case, one in which the total capacity shall not exceed 50 per cent., the quantity of air admitted in ordinary respiration shall not be lessened, in by any means the same proportion; a circumstance which, conjoined with the immense destruction of the air-cells, seems to imply, that not only do the bronchi contain air, but that some of them have the power of expelling and receiving some portion of it. But however this may be, it is clear that as the air-cells of a lobule lose their power of action from a deposit of tubercle, the smallest bronchi must become implicated also.

Thus as the function of the lungs is performed by the air-cells exclusively, any diminution of that process must be directly due to a diminished action of those cells. I have ascertained that the action of the cells consists in the acts of extension and retraction in a spiral manner, and it is probable that the first evidence of the lessened action of the cells would be found in the imperfection of the mode of elongation.

Thus I have endeavoured to show that the earliest indication of phthisis is the lessened action of the air-cells, and it is highly probable that this is commonly associated with or resulting from depressing agencies. We all know in our own persons the temporary effect of depressing causes, as grief, or disquietude of any kind over the action of the lungs, and have observed the slow and shallow respiration followed by long sighs or yawns possibly indicating the accumulation of carbonic acid in the air-cells of the lung from the previous incomplete respiration. Moreover, as the function of respiration is so important and so unceasing, and as the air-cells are of a delicate organization, it is quite clear that they must be highly endowed with nervous influence from the cerebro-spinal, excito-motory, and sympathetic systems, and, consequently, be extremely liable to their own special diseases. As they have the power of extension and retraction, that power may be modified both towards decrease as well as increase; and as they are of delicate organization and so endowed with nervous influence, it is certain that they will both sympathize greatly with the general nervous system and be greatly influenced by it, and also be liable to their own special diseases, arising more or less from local causes only. Thus, the lessened action which constitutes or leads to phthisis may be either general or local; and whilst admitting that the multiplicity of causes point to a general action, we must not ignore the possibility of their being due to local causes only. I am anxious to refer to this, for we have each of us seen cases which appeared to us to have arisen from some local cause, as some prior local disease; but the prevailing notion of the general and blood origin of phthisis has led us to throw doubts over the correctness of our belief. Without venturing an assertion on a subject about which so little is known, I would take courage to ask if there are not grounds for the belief that acute phthisis is the local, as contrasted with the chronic phthisis, which is the more general form of the disease. Upon this point, however, I would not dwell, but I feel convinced that a wide field for inquiry is yet uncultivated in the diseases of the air-cells of the lungs, and that whilst we may not have given too much attention to the mere conduits of the air, we have given too little to the all-important air-vesicle.

That the lessened action of the air-cells to which I have referred as an evidence of phthisis is exclusively restricted to that disease I dare not venture to affirm. In many cases of chronic bronchitis there is lessened vesicular murmur and some lessened resonance on percussion, both due, in some degree, to the same causes as in phthisis. Whenever, however, there is lessened vesicular murmur with lessened mobility and both flattening and atrophy, and at that early period short and feeble inspiration with lessened quantity of admitted air, and with or without the slight dulness referred to, we may safely affirm that to be a case prone to tubercle.

At this period there is no deposition of tubercle, and it is that which I have ventured to characterize as the first or pretubercular stage, whilst the presence of tubercle should characterize the second, and that of destruction of the lung the third stage of the disease. It is, however, quite understood that as the whole of the lung is not implicated at once, the disease will have reached its third stage in certain portions, and only the first stage in others. The first stage probably endures for a lengthened period in many cases, for in how many instances have we noticed persons, evidently prone to this disease, resist its attacks for years. But whether it endures for many years or not, it is sufficiently long to permit of the full employment of remedies fitted to restore the lessened functions. The difficulty lies not in the duration of the stage, but in the absence of agreement in the profession as to the existence, signs, and importance of this early condition, and more particularly from the ignorance of the public on this point, whereby the stage has long passed away before advice is sought. If, however, the profession could agree on this point, it could readily direct the attention of the community to it, and by advising a frequent examination of the chest of the younger members of families, obtain opportunities of detecting the disease thus early. Moreover, a belief in the hopeless nature of the disease, when once fairly set in, would induce the public to turn with readiness to the prevention of, or to the treatment of it in its earlier moments, if they fully understood that there is a conquerable stage in the disease in which treatment would commonly succeed.

Assuming, then, that the theory of the nature and production of phthisis now advanced is a true one, I proceed to offer an explanation of a few attending circumstances of interest.

The air which is chiefly involved in this lessened action of the air-cells is the residual air, or that which cannot be expelled by any voluntary effort during life. This is evident from the fact that the patency of the air-cells is maintained by the presence of this residual air, and that with lessened action, or closure of the air-cells, there must be a lessened quantity of the residual air. That the tidal air, or that which enters and leaves at each respiration, is not materially implicated, is evident from the small quantity of that as compared with the residual air, and from the fact that the former may remain intact whilst four-fifths of the latter may have been lost. The interest of this question is in relation to the value of the spirometer as evidence both of decrease and of improvement, and especially in relation to the chemical changes of the air in the lungs which are mainly or exclusively restricted to the residual air. Thus, no great diminution of the action of the air-cells can occur without a diminution of the residual air, nor any lessening of the latter without a lessening of the chemical and vital effects of respiration.

The explanation of the localization of tubercles to the apex of the lung, in the early stage, may be thus explained: As the direction of the trachea and the divisions of the bronchi is downwards, as is also the chief inspiratory power, it follows that the lower parts of the lung will be more readily and perfectly filled than the upper, and that the full distension of the air-cells at the apex can only follow the full distension of these at the base, whereby the distended base, as a fulcrum, directs the full current to the apex. This may also be proved by any one taking in a very full inspiration, for he will not perceive the peculiar sensation of distension at the apex until the inspiratory effect has reached its utmost extent. Therefore at all times the extension of the cells of the apex will be proportionably less than elsewhere, and in a state of disease with enfeebled inspiratory power, the disparity will be still greater. Hence, in the first stage of phthisis, the part first affected will be the apex.

For the same reasons the liability to morbid actions will be greater there than elsewhere, for with lessened action there will be lessened vital energy, and consequently less power of resistance to morbid changes. Moreover, with less action there will be a lessened circulation, for nothing is more certain than that the circulation through the lungs is mainly supported by the act of respiration, and consequently as the latter fails so will the former, and this will, of course, be to a greater extent if solid matter is deposited so as to impede the passage of air through the smaller bronchi. Then with the lessened circulation there will be an increasing tendency to disease and a lessened power of absorption, whereby morbid or natural products may be less perfectly and quickly removed.

The air-cells having thus lost a part or the whole of their vital powers, they are fitted to become the depositories of diseased material, and hence, on a well-known principle, we may account for the selection (so to speak) of the lungs by tubercle, on the ground, amongst others, of the vast mass of such fitter receptacles which that organ offers.

To determine the origin of tubercle, and the immediate cause of its deposition in the air-cells, is a matter of great difficulty; but I venture to call your attention to the assertion of Dr. Carswell, that tubercle is first found on the free surface of the air-cells, and to that of W. Addison, viz., that tubercle on the lung is transformed epithelium. So long as the existence of epithelium in the air-cells was denied, this latter opinion was fairly open to question, but now that we must admit its existence, I feel that there are very high probabilities in favour of Dr. Addison's theory. No one who has carefully examined the material found in the air-cells in acute phthisis, or even in favourable circumstances in the recent formation in chronic phthisis, can fail to be struck with the great resemblance which exists between that structure and the epithelium of the air-cells, and the transformations which occur in them are common to both. It would be unjust to contrast them at a later stage, for such retained epithelium must be unnatural and diseased, and hence very fitted to put on further degeneration, and by that process to lose its natural characters, and to gain the earthy and oily products which constitute tubercle in its advanced condition. Moreover, on this theory the explanation is simple and natural, for as the air-cells lose their power of action and vital energy, the epithelium will be retained in the cell, and in its formation will be less and less perfect. Hence, after a time, in one or many cells, and in one or many lobules, the cells will have become filled by the accumulated epithelium, and when the distension is so great as to overcome the resistance of the lobular membrane, it will press upon adjoining lobules and impede their action, and thus in an increasing ratio increase the liability to disease. In this mode we readily account for the greater comparative rapidity of phthisis after, than before, the deposit of tubercle, and the rate will doubtless be increased or lessened as the vital power of resistance of the lobular membrane, and therefore of the system as a whole, is great or little.

I do not think that frequency of co-ordinate deposits of tubercle elsewhere is adverse to this theory, for, assuming that the deposits are in many instances identical, they are commonly found in the same situation in different organs, viz., the epithelial cell, and whatever may lead to the diseased action in one organ may lead to the same in the same structure in another organ. Moreover, as we are not agreed as to what we shall call tubercle, it is a reasonable doubt which would question the identity of deposits in different structures; and then, again, as two morbid actions may be concurrent and not necessarily resulting one from the other, but both possibly dependent upon one or diverse anterior conditions, it is quite possible that a deposit may occur in the lungs and at the same time elsewhere, and both be due to the concurring action of depressed general vital energies acting separately in different localities.

We must admit that in many instances scrofulous deposits in various parts of the body do occur with pulmonary phthisis, but on the other hand, I am able to prove by long inquiry at the hospital, that the proportion of phthisical persons in whom, or in whose families, external evidences of scrofula have existed, is extremely small, and I feel justified in affirming the occurrence of the two phenomena to be co-ordinate action only. If the blood be diseased or scrofulous, and fitted to pour out morbid products, what can be so likely a cause of phthisis as the general exhaustion and variableness of system which accompanies it? or, on the other hand, in cases of phthisis with original defect of constitution, what so likely a result as that the blood shall become diseased as the phthisis advances, and lead to that state which, without the phthisis, is so often found.

I am anxious to make a distinction between circumstances which may be co-ordinate, or even dependent more or less on each other in certain instances, and those which are identical either in their nature or results. I believe that the connection of phthisis with scrofula is that of a coincidence, and is not evidence of any necessary relationship between the two diseases.

On the above principles we may also account for the intractable nature of the disease; for besides that it is located in an organ essential to life, its tendency is ever to the increase of morbid products, and its power, both of resisting morbid agencies and of removing morbid products, becomes less and less.

The occurrences of hæmoptysis at a period anterior to the deposit of tubercle, *à fortiori* at a period before the occurrence of destruction of tissue, is thus readily explained. As the action of the air-cells is in any part lessened, so will the quantity of blood in that part diminish, and as the total quantity of blood in the body will probably remain much the same, there must be a larger quantity in other parts of the lung, and hence with congestion in the immediate locality of a diseased cell, a lobule, or elsewhere, there is a proneness to hemorrhage, and the exciting cause, cough, is only required to give development to it. In order to meet this difficulty nature increases the frequency of both respiration and pulsation, and thus for a time maintains a balance between the pulmonic and systemic circulation; but a period must arrive when that expedient will fail. If this be a fair explanation, it becomes a very important inquiry as to the propriety of interfering with such an hæmoptysis, or any increased frequency in the functions, and as to the possibility of arresting the former through the general circulation.

These explanations might be carried to any length, but I have already occupied too much space. My aim is rather to ask my professional brethren to draw their attention away from opinions, in reference to this great disease, which are both unprofitable and tend to stop further inquiry, and to direct them so that a rational treatment may be adopted, and a field laid well open to investigation. To regard phthisis as a blood disease is simply to shroud it in obscurity, and foster the belief, too well grounded it must be confessed, of the incurability of the disease; but the views which I have ventured to offer, seek to attract attention to the prevention of the disease, and to its very early arrest when it has commenced, and afford promise that in both directions our efforts will be successful.

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Art. VIII.—*Case of Wound of the Abdominal Walls, with Protruded Omentum.* By Walter F. Atlee, M. D., one of the Surgeons to St. Joseph's Hospital.

The following case of *wound of the abdominal walls, with protruded omentum,* it is believed, will be found both interesting and instructive. Cases of the kind are not frequent, and surgical authorities differ widely in regard to their treatment. The account of the case is drawn up from the notes taken daily by the Resident Physician, Dr. J. J. Cruice; and it is but proper to premise that it was always determined to report it, whatever might be its termination:—

Daniel Blaney, æt. 22, of medium size, robust and healthy, a private in Company A, 21st Pennsylvania Regiment, was admitted into St. Joseph's Hospital, September 31, 1861. He had received, six hours before admission, while in a state of intoxication, a stab from a knife, in the abdomen. There was constant vomiting, with coldness of the extremities, and a small, frequent pulse.